A Deterministic Model for Measles
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A deterministic model of recurrent epidemics is con-
structed using a non linear relationship between infection
rate and number of contacts. Epidemic waves which are not
damped are predicted and a relationship between commu-
nity size and the period of recurrence is established. A pos-
sible explanation of measles outbreaks is suggested.

Introduction

The theory of periodic outbreaks of diseases such
as measles dates from a paper by Soper [1]. This
model is inadequate because the periodic epidemic
waves are invariably damped down to an equilibrium
level of infectives. Moreover the Soper model does
not predict the observed relations between community
size and periodic time: it is observed that epidem­
ics tend to die out or have long periods of recur­
rence in small communities. To remedy these defects
it is generally held that a stochastic approach is re­
quired (see Bartlett [2,3] and Black [4]). In this paper
a deterministic mechanism is described whereby re-
current epidemics may be generated and by which
smaller communities are expected to have less fre-
quent epidemics.

The Model

Let S denote the effective number of susceptibles.
In the case of measles this will be some subset of
the immature section of the society. Very young children
below school age and mature adults rarely suffer
from measles. Let I denote the number of infective
persons. Let C denote the number of persons who
although recovered from the disease have not yet
reached maturity (say 15 years old). Let R denote
the number of mature persons (who may or may not
have had the disease while immature) who are im-
une and whose number controls the supply of new
susceptibles through the birth rate. The basic equa-
tions of the model are

\[ S = -\lambda (SI)^p - \tau_1 S + \alpha R \]
\[ I = \lambda (SI)^p - \mu I \]
\[ C = \mu I - \tau_2 C \]
\[ R = \tau_1 S + \tau_2 C - \alpha R \]

where \( \lambda, \mu, \tau_1, \tau_2, \mu \) and \( \alpha \) are positive parameters.
Here the total population is constant in size and, for
simplicity, has a conserved age structure i.e.

\[ R = 0, \quad S = I + C = Y_0 \]

where \( Y_0 \) is a constant.

When \( p = 1 \) the model does not differ significantly
from the Lotka-Soper model [5].

Equilibrium will occur when \( \lambda (SI)^p = \tau_2 C = \mu I \).
We shall seek oscillatory solutions by setting

\[ S = S_0 + \epsilon_1, \quad I = I_0 + \epsilon_2, \quad C = C_0 + \epsilon_3 \]

and neglecting powers and products of

\[ \epsilon_1, \epsilon_2, \epsilon_3 \]

\[ S_0, I_0, C_0 \]

where \( S_0, I_0 \) and \( C_0 \) are the equilibrium values of \( S, I \) and \( C \).

Solutions of periodic time \( 2\pi/\omega \) are possible with

\[ \omega = \frac{1}{2} \sqrt{P} \]

provided

\[ P = 4\mu^2\tau_2 - (\mu p (1 - r) - \mu + \tau_2)^2 > 0 \]

where \( r = I_0/S_0 \).

Solutions will contain a factor

\[ \exp(Q/2) \]

where

\[ Q = \mu p (1 - r) - \mu + \tau_2 \]

and will be damped if \( Q < 0 \), amplified if \( Q > 0 \) and
purely oscillatory only if \( Q = 0 \).

When \( p \equiv 1 \) \( Q \) is necessarily negative which is why
the original Soper model was discarded. When \( p > 1 \),
however, the solutions oscillate and grow in amplitu-
de so that the approximation by means of which
they were derived will eventually become invalid.

We suggest that the values of \( p \) and \( \lambda \) should be
fixed in such a way that amplification occurs and ex-
tinction becomes the likely consequence of the unap-
proximated equations. The recurrence of the disease

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and hence the period will depend on the mechanism of re-introduction and a possible model is now described.

We propose that visitations from outside the community by human hosts of the disease are more likely the larger its size and we shall assume that the mean number of infectives visiting the community is proportional to its size $N$. The modified model is then, on eliminating $R$

\[
S = -\lambda S^p (I + \varepsilon N)^p + \tau_2 C
\]

\[
I = \lambda S^p (I + \varepsilon N)^p - \mu I
\]

\[
C = \mu I - \tau_2 C
\]

where $\varepsilon N$ is the number of visiting infectives. If $S \to 0$ and causes the disease to die out the susceptible population will grow and the reintroduction term will restart the epidemic oscillation. The smaller $\varepsilon N$ the longer the periodic time which results. We may have a situation in which $I \to 0$ without $S \to 0$ and then the fadeout of disease may persist.

### Feasibility Calculations

We shall assume that the model relates to a typical sub-population of 1,000 persons with $Y_0 = 200$. The calculations have been made using a simple finite difference scheme in which the time step is 0.1 years. We have assumed that an infective has a lifetime of about one third of a time step (about 12 days) and accordingly have set the cure parameter $\mu = 0.75$. The transfer parameter has been fixed at $\tau_2 = 0.01$ (100 time steps from age 5 to 15 years). The factor $\varepsilon N$ has been fixed at 0.05 (Table I) which represents a mean visitation rate of infectives of 10 per 0.1 years per 200,000 total population. Table II shows the effect of reducing $\varepsilon N$ to 0.005 when $\lambda = 0.02$ and $p = 1.5$ i. e. to 1 infected person per 0.1 year per 200,000.

For purposes of gauging when an epidemic occurs an arbitrary level of 1 infective per 0.1 years per 1,000 has been set.

It is felt that the introduction of the parameter $p$ provides a new feature in the deterministic modelling of epidemics and offers an explanation of recurrent outbreaks whose periodicity depends on population size. More sophisticated models can easily be envisaged e. g. an infection term

\[
\sum_{i=1}^{n} \lambda_i (S(I))^p_i
\]

could be treated but there is little indication from our calculations that additional parameters will be necessary.